sclerosis) and motor unit disorders (amyotrophic lateral sclerosis, progressive bulbar palsy, muscular dystrophy, polymyositis or dermatomyositis, and myasthenia gravis). Symptoms of oropharyngeal dysphagia do not differ greatly in these various diseases. Dysphagia occurs with liquids and solids, and multiple attempts may be necessary to swallow successfully. Nasal and oral regurgitation often occur immediately after a swallowing attempt, sometimes with a forceful spraying of the mouth contents. Tracheal aspiration is common, possibly leading to pneumonia. In severe cases, patients may not even be able to swallow their own saliva, and malnutrition and weight loss may result.[4]

In esophageal dysphagia, the swallowing act is initiated normally, but a bolus of food fails to progress into the stomach. Patients typically describe a feeling of food sticking during swallowing. This sensation is generally in the esophagus but may be referred proximally to the neck, even with a distal esophageal lesion.[5] The differential diagnosis of esophageal dysphagia includes obstructions (rings and webs, benign strictures, and cancers) and motility problems (achalasia, scleroderma). Generally, obstructive disorders (unless severe) cause dysphagia for solids only, whereas motility disorders cause dysphagia for both liquids and solids. Heartburn is typical of scleroderma and strictures. Cancers cause a rapid progression of symptoms, and rings and webs usually cause intermittent dysphagia. Oral regurgitation, but not nasal regurgitation, may occur with esophageal dysphagia, generally hours after swallowing.[4]

Careful questioning can help to determine the cause of dysphagia. If the history suggests myasthenia gravis, an edrophonium test should be done. Although no precise figures have been published, sensitivity is estimated to be about 86% in the ocular form of the disease and 95% in generalized myasthenia—not limited to ocular symptoms. Specificity for the edrophonium test is not clear-cut, but a number of diseases are known to produce false-positive results, including amyotrophic lateral sclerosis and the Guillain-Barré syndrome. A serum acetylcholine-receptor antibody titer is the appropriate second-line confirmatory test, because specificity is high. Sensitivities of 64% (ocular) and 89% (generalized) have been reported.[6] These two tests are accurate and inexpensive enough that they should be done in almost every case of suspected myasthenia gravis.[6,7] Electromyographic techniques (conventional or single-fiber) can also be used to confirm the diagnosis. These techniques have a high specificity, and single-fiber electromyography is fairly sensitive but not widely available. Therapy includes both medical and surgical modalities, and established guidelines are available.[8-10]

In summary, myasthenia gravis is an often-overlooked cause of dysphagia. The differential diagnosis of dysphagia includes oropharyngeal and esophageal causes. When the history suggests oropharyngeal dysphagia, neurologic causes, including myasthenia gravis, must be carefully considered. A directed neurologic history and physical examination, possibly including a nighttime examination, is necessary and sufficient to diagnose most cases of this disorder.

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Nontraumatic Splenic Hematoma Related to Cocaine Abuse

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SPLENIC HEMATOMA and rupture are most commonly seen after blunt abdominal trauma. In rare instances splenic hematomas can occur without trauma, usually in patients with splenomegaly or underlying hematologic disorders.[1,2] This is the first report of a case of an otherwise healthy man in whom a splenic hematoma developed shortly after he used cocaine intranasally.

Report of a Case

The patient, a 40-year-old man who habitually used cocaine, presented to a local emergency department with left upper quadrant abdominal pain radiating to his left shoulder. Seven hours previously, he had "snorted" an unknown quantity of cocaine. He was working on his automobile when the pain began and became progressively severe. In the emergency department, his anterior chest wall was sensitive, but his lungs were clear, heart regular, and an abdominal examination revealed good bowel sounds. His abdominal wall was soft, without

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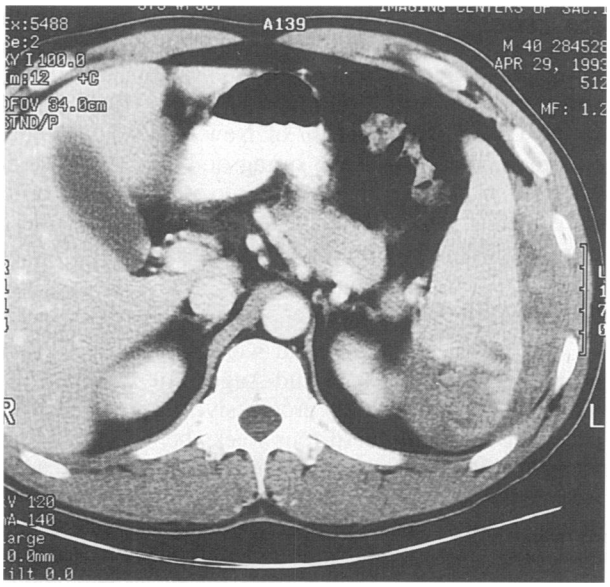

Figure 1.—A computed tomographic scan of the abdomen with contrast shows a subcapsular splenic hematoma and a tear in the splenic parenchyma.

tenderness. Laboratory studies done in the emergency department showed a leukocyte count of 16.9×10^9 per liter (16,900 per mm^3) and a hemoglobin level of 130 grams per liter (13.0 grams per dl). His chest radiograph and electrocardiogram were normal. After determining that a myocardial infarction was unlikely, the emergency department physician sent him home. He was admitted to the hospital a few days later after his private physician diagnosed a splenic hematoma by ultrasonogram.

On examination the patient was afebrile and normotensive. He had tenderness in the left lower abdomen, but his bowel sounds were normal and there was no hepatosplenomegaly, lymphadenopathy, or heart murmur. His left testicle was tender and swollen, but the rest of the examination showed no abnormalities. His leukocyte count was 7.9×10^9 per liter (7,900 per mm^3), a hemoglobin level was 107 grams per liter (10.7 grams per dl), and a platelet count was 172×10^9 per liter (172,000 per mm^3). His prothrombin time was 13 seconds, and a partial thromboplastin time 28.5 seconds. His computed tomographic (CT) scan (Figure 1) showed a splenic hematoma and a possible intrasplenic tear.

The patient's blood count was monitored and remained stable. After a few days of observation, he was discharged home. On follow-up two months later, he had remained drug-free, and his blood count had returned to normal. His testicular swelling and tenderness cleared after treatment with trimethoprim and sulfamethoxazole. A urologist felt that his epididymitis was unrelated to his splenic tear and found no evidence for retroperitoneal dissection of the hematoma. A follow-up CT scan showed almost complete resolution of the splenic hematoma.

Discussion

Cocaine abuse is epidemic and associated with a variety of medical complications.[3,4] This is the first report of atraumatic splenic hemorrhage occurring after cocaine abuse. There was no underlying systemic infection, coagulopathy, or hematologic disorder.

A case has been reported of a woman with the sickle cell trait with infarction of part of her spleen occurring a few hours after she used cocaine intravenously.[5] Conceivably the patient in this report may have first had infarction of a portion of his spleen, then hemorrhage into this area when cocaine-induced vasospasm resolved. Hypodense areas in the spleen could have represented other areas of infarction. The delay in symptoms supports this mechanism because primary hemorrhage (as with intracerebral hemorrhage from cocaine) is usually immediate, but myocardial infarction often occurs hours after ingestion.[6,7] The effects of cocaine on platelet aggregation may be important in both splenic and myocardial infarction.[8-10] The spleen is susceptible to infarction. Other possible mechanisms for his hematoma included arteriolar rupture due to cocaine-induced hypertension or splenic traction while he bent over his car to work on the engine.

Splenic hemorrhage is important to recognize because it can lead to hemorrhagic shock and death. Presenting with left-sided upper abdominal pain radiating to the left shoulder, these patients are often examined for myocardial ischemia. Indeed, this patient was sent home from the emergency department after a cardiac cause for his pain was excluded. With recent reports of cardiac and noncardiac chest pain in cocaine users presenting to emergency departments,[11,12] clinicians may overlook the possibility of splenic injury. If thrombolytic therapy is instituted, an iatrogenic disaster may occur, as this type of therapy in itself is a rare cause of splenic hematoma.[13] Clinicians should therefore include splenic hemorrhage in their differential diagnosis when seeing cocaine-using patients with chest, left upper quadrant, or left shoulder discomfort. Chest pain radiating to the left shoulder is a classic sign of subdiaphragmatic irritation, and a CT scan or ultrasonogram should be strongly considered, even in the absence of pronounced abdominal findings, once cardiac or intrathoracic disease is excluded.

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Hantavirus Infection Following Wilderness Camping in Washington State and Northeastern California

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THE HANTAVIRUS PULMONARY SYNDROME is a newly recognized infectious disease with a high case-fatality rate. Initially described in the Southwest, cases of the disease have now been reported from a wide geographic area in the United States.[1] The clinical consequences of this infection are severe: Of the first 17 cases reported, 50% of the patients presented with hypotension, and rapidly progressive pulmonary edema developed in 15 (88%).[2] As of June 1995, 60 of the 113 patients diagnosed with the hantavirus pulmonary syndrome (53%) have died.[3] The etiologic agent is a previously unrecognized hantavirus, a single-stranded RNA virus that belongs to the Bunyaviridae family.[4-7] Although a uniform nomenclature has not been consistently used to describe this virus, the currently accepted name is *Sin Nombre* virus (Ali S. Kahn, Special Pathogens Branch, Centers for Disease Control and Prevention [CDC], oral communication, July 1, 1995).[1,5,6,8] The primary mode of transmission suggested by epidemiologic studies has been exposure to rodent excreta in and around rural households.[1,9]

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In this report, we describe the case of a patient with the hantavirus pulmonary syndrome who presented to a northern California hospital in the summer of 1992, before the cluster of cases of the syndrome that occurred in the Four Corners region of New Mexico, Arizona, Colorado, and Utah in the spring of 1993. This case is remarkable in that it is the first reported case likely to have been acquired through exposure during wilderness camping in Washington State or northeast California.

Report of a Case

The patient, a 49-year-old woman, was seen in an emergency department in mid-August 1992 with a flu-like syndrome and rapidly progressive dyspnea. She had been in good health until four weeks before admission, when, while she was camping in North Cascades National Park in Washington State, several skin lesions developed on her anterior lower extremities that she attributed to insect bites. She described them as 3 cm in diameter, round, intensely pruritic, raised, erythematous, with a central white area, and resolving within a week.

Two and a half weeks before admission, she had generalized weakness, crampy abdominal pain, nausea, anorexia, and foul-smelling loose stools. These symptoms continued but waxed and waned in intensity. Eight days before admission, she had fever, chills, and myalgias and arthralgias involving the hands, shoulders, elbows, and knees. Subsequently, she had a frontal headache associated with fever to 39.4°C (103°F) and worsening joint pain. Two days before admission, she was short of breath and had a nonproductive cough and persistent fever. The dyspnea progressed until the morning of admission, when she felt she "could not breathe."

About 4½ weeks preceding her illness, she also had camped for three days in the Mono Lake region in the eastern Sierra of California (Figure 1). During both camping excursions, she filtered her water and brought her own food because of "food allergies," storing it separately in her tent. She slept on the ground outdoors or in partially open tents. There were no reported rodent contacts, and no other camper who accompanied her became ill following either trip.

Her medical history was unremarkable; she specifically had no history of immunosuppression, diabetes mellitus, or lung disease. The patient resided in an urban dwelling in Oakland, California, owned a healthy dog, and did not report rodent exposures at her home to her friends or relatives. She worked as a clinical psychologist at a local hospital and was physically active, running as many as 32 km (20 mi) weekly. She did not smoke, drink alcohol, or inject drugs.

On admission, she was in obvious respiratory distress and had a fever of 38°C (100.4°F), a blood pressure of 70/50 mm of mercury, a heart rate of 102 beats per minute, and a respiratory rate of 32 breaths per minute. She was alert, and her head and neck were normal. Her skin had scattered hyperpigmented areas on the lower extremities. Examination of her lungs revealed rales at